AD-A128 180 EVOKED POTENTIAL STUDIES OF CENTRAL NERVOUS SYSTEM INJURY DUE TO IMPACT ACCELERATION(U) TEXAS RESEARCH INST OF MENTAL SCIENCES HOUSTON B SALTZBERG ET AL. 1977 N00014-76-C-0911 1/1

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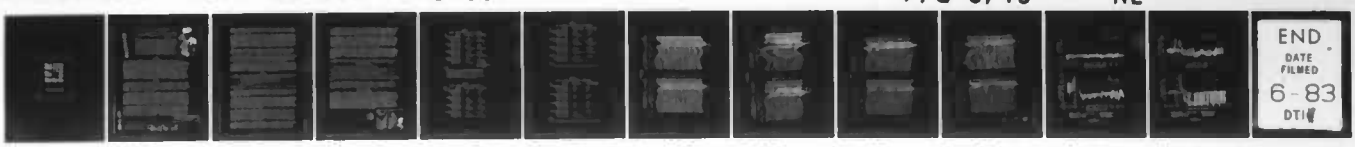

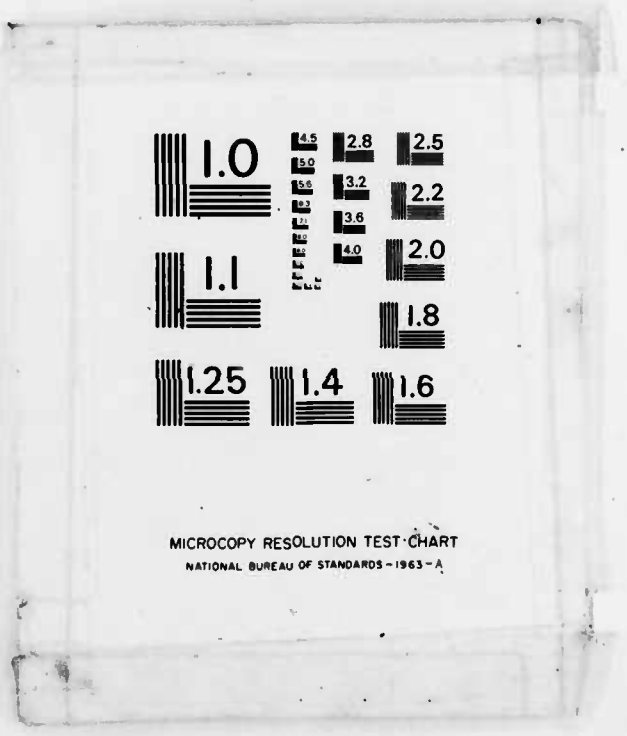
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MICROCOPY RESOLUTION TEST CHART
NATIONAL BUREAU OF STANDARDS - 1963 - A

EVOKED POTENTIAL STUDIES OF CENTRAL NERVOUS SYSTEM INJURY DUE TO IMPACT ACCELERATION*

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1977

This paper reports on one aspect of a comprehensive program designed to investigate the effects of various levels of impact acceleration on the functional integrity of the nervous system. The results described are based on the measurement of afferent neural transmission in the Rhesus monkey as revealed by latency and amplitude changes in the evoked potential (EP). In order to track the time course of recovery of latency and amplitude with high time resolution, automated methods for detecting peak amplitude and latency of components of the evoked potential were developed. These methods were applied to EP data recorded during impact experiments on Rhesus monkeys.

PREFACE

Because of the growing concern for the human and economic cost of vehicular accidents, studies to systematically evaluate the physiological effects of impact acceleration on the head and body are becoming increasingly urgent. Most research in this problem area has been limited in scope because of the complexity of the physiological measurements and the extensive technological resources required to achieve precise recording and control of the large number of mechanical and biological variables involved. In response to this need, a comprehensive program has been developed at the Naval Biodynamics Laboratory (NBDL) for acquiring the fundamental data needed in the development of improved measures to reduce injuries caused by impact accidents. The NBDL facility is equipped with an integrated battery of scientific instrumentation and computers to collect and correlate anatomical, physiological, neurological, radiological, and mechanical data. This paper presents the initial analysis of an electrophysiological data base which is currently being examined and correlated with other physiological and physical measures by specialists in neurophysiology and neuroanatomy at NBDL. The major objective of this continuing analysis is to interpret and model the results of the experiments at NBDL.

METHODS

Four Rhesus monkeys were subjected to a total of eight sled impact acceleration runs at NBDL to reproduce the dynamic forces which act on the head, and on the spinal column and cord in a lateral (-Y) collision. Each animal was subjected to a 10-G control impact, followed later the same day by a larger impact. The larger impacts were: 30-G for animal AR-8849, 50-G for animal AR-2152, 70-G for animal AR-8695, and 90-G for animal AR-8816. Analyses of only the 30, 50, 70, and 90-G runs are presented here since the 10-G runs showed no significant post-impact EP changes.

Electrical stimulation was applied to the spinal cord with recording of evoked activity from the Left and Right Sensory-Motor Cortex (CXL and CXR). Surgical procedures for electrode implantation were carried out under barbituate anesthesia with endotrachial intubation and atropine premedication. Stimulating electrodes were a five-in-line lead parallel array placed over the spinal cord. Recording electrodes were placed over the left and right sensory-motor cortex. Details of the electrode configurations and surgical implantation procedures are described in Reference 1 (P.R. Walsh, et al., "Experimental methods for evaluating spinal cord injury during impact acceleration"). All stimuli were constant current rectangular pulses of 0.2 millisecond duration. Current levels (approximately 1.25 milliampere) were applied sufficient to obtain good afferent evoked potentials.

Copies of the analog data tapes from the NBDL -Y impact experiments were processed at the Texas Research Institute of Mental Sciences (TRIMS) using Average Evoked Potential (AEP) analysis programs written specifically for this project. The analog data consisted of two channels of EEG data, a stimulus marker channel, and a time-code channel. These data constituted the input to a PDP-11 computer equipped with an AR-11 analog-to-digital converter (10-bit resolution). The time code was used to control the digitizing start and stop times relative to experimental impact. The stimulus marker controlled the start of data acquisition for individual responses. In order to achieve high resolution in

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measuring the latency of AEP components, the analog tape was slowed to half its normal speed, and appropriate adjustments were made to playback discriminators and the sampling interval. The final digitized data resolution was 25 microseconds per point (equivalent to 40,000 samples per second).

Starting on the rise of the stimulus mark pulse, 2000 digital samples were used to obtain AEPs of 50 milliseconds duration. Initially, 5 individual responses were averaged to create each AEP. The AEPs were written to digital tape for subsequent processing. Preliminary examination of the AEPs (based on 5 responses) immediately following impact revealed a significantly noisy pattern and, therefore, additional averaging was necessary. However, to achieve good time resolution of temporal changes in amplitude and latency of components of the AEPs, it was necessary to minimize (within the constraints of noise) the number of individual responses used to obtain a smooth AEP. Using AEPs consisting of 50 individual responses met both criteria in that the resulting improvement in signal-to-noise ratio gave a smooth AEP while providing a reasonably good time resolution of 10 seconds.

In order to visualize overall changes in AEP waveshape, compressed AEP plots were produced. These plots show the time course of AEPs over a period of 12 minutes, beginning 2 minutes prior to impact. Compressed AEP plots for the Left and Right Cortical Leads (CXL and CXR) from each of the 4 experiments are shown in Figures 1 through 4.

The feature most common to AEPs from the 4 different animals was a peak which occurred in the latency range from 9 to 13 milliseconds following the stimulus. Except in animal AR-2152, used for the 70-G run, this peak was positive-going, and will be referred to as E10. An AEP component in the latency range 15 to 20 milliseconds (designated E15) was found in all animals except AR-8816, the animal used for the 90-G experiment.

Quantification of changes in the AEPs was done by tracking the amplitude and latency of the E10 and, where possible, the E15 peaks. The mean and standard deviation of the measures were computed from 23 AEPs, starting 4 minutes prior to impact. These were used in comparing pre- and post-impact AEP measures. Changes were defined as significant when the measured value for 2 successive AEPs deviated by more than 1 standard deviation from the pre-impact mean. Recovery time for a measure was defined as the time from impact to the first value within 1 standard deviation of the pre-impact mean.

RESULTS

Tables 1 through 4 summarize effects on the amplitude and latency of the E10 and E15 AEP components from the 4 acceleration levels studied. Listed in the tables are:

1. The percent relative deviation of the measure during 4 minutes pre-impact (standard deviation ÷ mean × 100),
2. The maximum change post-impact, expressed as percent of the pre-impact mean, and
3. The recovery time

For those instances where the post-impact changes were not significant, the recovery time is reported as zero.

Table 1 shows the effects of impact on the amplitude of the E10 component. At 30-G, the amplitude is reduced in both the CXR and CXL leads. Following 50-G acceleration, the amplitude is reduced more in the Right Lead (76%) than in the Left Lead (70%). The 70-G impact produced an <u>increase</u> in the amplitude of the E10 peak on the left side, and a <u>decrease</u> on the right side.

The largest and most asymmetric effect on amplitude took place at 90-G acceleration, as shown in Figure 5. The amplitude of the Left Lead E10 component increases slightly for 30 seconds following impact, while the positive-going E10 component is completely obliterated from the Right Lead. This effect lasts for 4 minutes post-impact. Between 4 and 5.9 minutes, the amplitude recovers to nearly its pre-impact value before falling again. The amplitude leaves the recovery band again at 6.8 minutes and reaches a reduced stable value by 10 minutes post-impact. Between 10 and 58 minutes, the amplitude exhibits a very slow recovery trend. The amplitude variability from AEP to AEP is markedly less during this time than during the pre-impact period.

For all 4 acceleration levels, the E10 amplitude recovery time recorded from the Right Lead is considerably longer than for the Left Lead. Recovery in the Left Lead following 50-G impact is slightly longer than it takes at 30-G. The Right Lead at 50-G recovers in about half the time compared to 30-G. At the 70-G acceleration, the Left Lead required 5 minutes to recover, while the Right Lead had not recovered during the 6.5 minutes of post-impact data studied. Only 6.5 minutes for this run were used due to a technical problem which is now being corrected. Two recovery times are listed for the 90-G run, the first (5.8 minutes) represents the initial amplitude rebound; the second (58.6 minutes) is for the long-term effect.

Table 2 is a summary of the latency changes for the E10 component of the AEP. There were no significant changes in latency associated with 30-G impact. At 50-G, the Left Lead component shows a 9.2% reduction in latency, while the Right Lead shows a 2.3% increase. The recovery time shown for the CXR lead of the 70-G run is not a reliable estimate because of the small maximum change relative to the pre-impact variability.

The 90-G acceleration gives rise to the most asymmetric effect. As shown in Figure 6, the latency of the Left Cortical E10 component increases 7.9% following impact, and recovery takes place within 1 minute. The left pathway latency increases and decreases again between 4 and 8 minutes post-impact. This time corresponds to the time when the Right Cortical AEP amplitude is rebounding. After 8 minutes, the latency of the left E10 component reaches a mean value which is about 2% less than its pre-impact value.

By comparison, the Right Cortical E10 component of the AEP reappears at 4 minutes post-impact, and its latency is 12% greater than before impact. Latency recovery takes about 7.2 minutes post-impact, a time which also corresponds with the amplitude rebound of this component. From 7.2 minutes on, the latency appears to stabilize to a slightly smaller value than it had pre-impact. This is probably due to the double hump shape of the E10 component in this experiment (Figure 4). Prior to impact, the second hump was consistently larger and was the one detected as the extremal. Following impact, the first hump is larger and, therefore, was detected as the extremal.

Table 3 summarizes the changes in amplitude of the E15 component of the AEP. The higher variability in the measured amplitudes during pre-impact time makes interpretation of this data more difficult. The 30-G impact had the effect of increasing the amplitude in the CXL lead, but had no effect on the CXR lead. At 50-G and 70-G, the amplitude on both sides was reduced, as was the amplitude of the E15 component in the CXL lead of the 90-G experiment. The E15 component could not be reliably detected in the CXR lead of the 90-G experiment. In all runs, recovery of amplitude occurred within 1.7 minutes post-impact.

Table 4 shows that only in the 90-G run is there any effect on the latency of the E15 component of the AEP. The latency increased by 7%, and recovered in 40 seconds.

SUMMARY OF FINDINGS

The most striking changes take place in the E10 component.

The recovery time of the E15 component varies directly with the impact intensity. Latency of the E15 component is only slightly affected by impact intensity.

At all 4 acceleration levels, the E10 amplitude component of the Right Cortical response takes longer to recover than the Left Cortical response.

At 70-G acceleration, there is a long-term effect on the E10 amplitude. This effect did not show up at lower impacts. This may be similar to the long-term effect present at 90-G.

The E10 component was obliterated from the CXR lead for 4 minutes post-impact at 90-G. However, small, earlier components remain clear for a considerable time after impact. These early components have latency and frequency characteristics similar to those recorded in human brainstem evoked response studies. At this G-level, these early components are affected quite differently than the E10 component.

CONCLUDING REMARKS

Insofar as lateral impact acceleration is concerned, our initial evaluation of the EP data produced in the NBDL experiments reported here indicates that neural propagation from the spinal cord to the sensory-motor cortex is more severely altered along the right pathway than along the left pathway. It should be emphasized that the analysis presented in this paper has been limited to an examination of only two components of the EP, a component at approximately 10 milliseconds latency and a component at approximately 15 milliseconds latency. There are other less prominent components in the range from 7 to 20 milliseconds which have not been analysed as yet, as well as late components which may have neurophysiological significance with regard to understanding the effects of impact acceleration on the motor nervous system. The early components in particular may offer some interesting insights on how brain stem activity is affected. As stated earlier, the electrophysiological results reported here are being integrated and analysed by NBDL in the context of its overall program of biodynamic measurement.

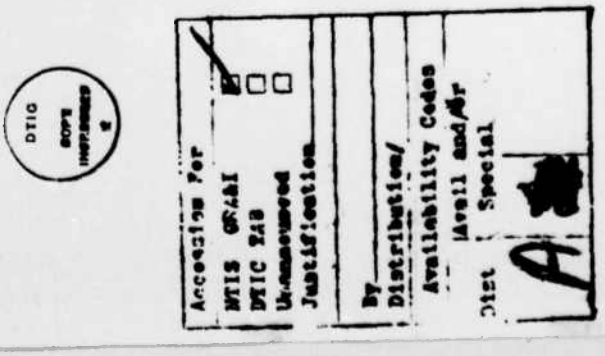


TABLE 1

SUMMARY OF CHANGES IN AMPLITUDE OF E10 COMPONENT

RUN	MEASURE	CXL-LEFT LEAD	CXR-RIGHT LEAD
30-G	pre-impact relative amplitude deviation	7.5%	6.6%
	Maximum change	-29.8%	-28.8%
	Recovery time	52 seconds	208 seconds
50-G	pre-impact relative amplitude deviation	15.9%	16.7%
	Maximum change	-69.8%	-75.9%
	Recovery time	69 seconds	100 seconds
70-G	pre-impact relative amplitude deviation	33.8%	23.0%
	Maximum change	55.9%	-52.0%
	Recovery time	300 seconds	390 seconds (Note 1)
90-G	pre-impact relative amplitude deviation	20.2%	15.5%
	Maximum change	29.9%	-100.0% (Note 2)
	Recovery time	30 seconds	356 seconds (5.9 minutes)
			3518 seconds (Note 3) (58.6 minutes)

NOTES

1. Data for only 6.5 minutes post-impact was tracked for the 70-G run. Recovery had not taken place by that time.
2. The positive E10 component was completely eliminated for 4 minutes post-impact.
3. After the 90-G impact, the amplitude recovered, then fell to a lower, slowly recovering value (see text).

TABLE 2

SUMMARY OF CHANGES IN LATENCY OF E10 COMPONENT

RUN	MEASURE	CXL-LEFT LEAD	CXR-RIGHT LEAD
30-G	pre-impact relative latency deviation	1.8%	1.0%
	Maximum change	NS	NS
	Recovery time	0	0
50-G	pre-impact relative latency deviation	2.2%	0.5%
	Maximum change	-9.2%	2.3%
	Recovery time	27 seconds	79 seconds
70-G	pre-impact relative latency deviation	0.5%	2.7%
	Maximum change	2.2%	4.4%
	Recovery time	94 seconds	21 seconds
90-G	pre-impact relative latency deviation	1.9%	1.6%
	Maximum change	7.9%	12.0%
	Recovery time	50 seconds	430 seconds

TABLE 3

SUMMARY OF CHANGES IN AMPLITUDE OF E15 COMPONENT

RUN	MEASURE	CXL-LEFT LEAD	CXR-RIGHT LEAD
30-G	pre-impact relative amplitude deviation	47.7%	65.7%
	Maximum change	119.5%	NS
	Recovery time	41 seconds	0
50-G	pre-impact relative amplitude deviation	24.6%	54.7%
	Maximum change	-78.7%	-100.0%
	Recovery time	100 seconds	69 seconds
70-G	pre-impact relative amplitude deviation	31.4%	20.4%
	Maximum change	-99.4%	-74.0%
	Recovery time	94 seconds	94 seconds
90-G	pre-impact relative amplitude deviation	30.0%	Note 1
	Maximum change	-69.0%	
	Recovery time	60 seconds	

NOTE

1. The E15 component could not be reliably tracked in the 90-G run.

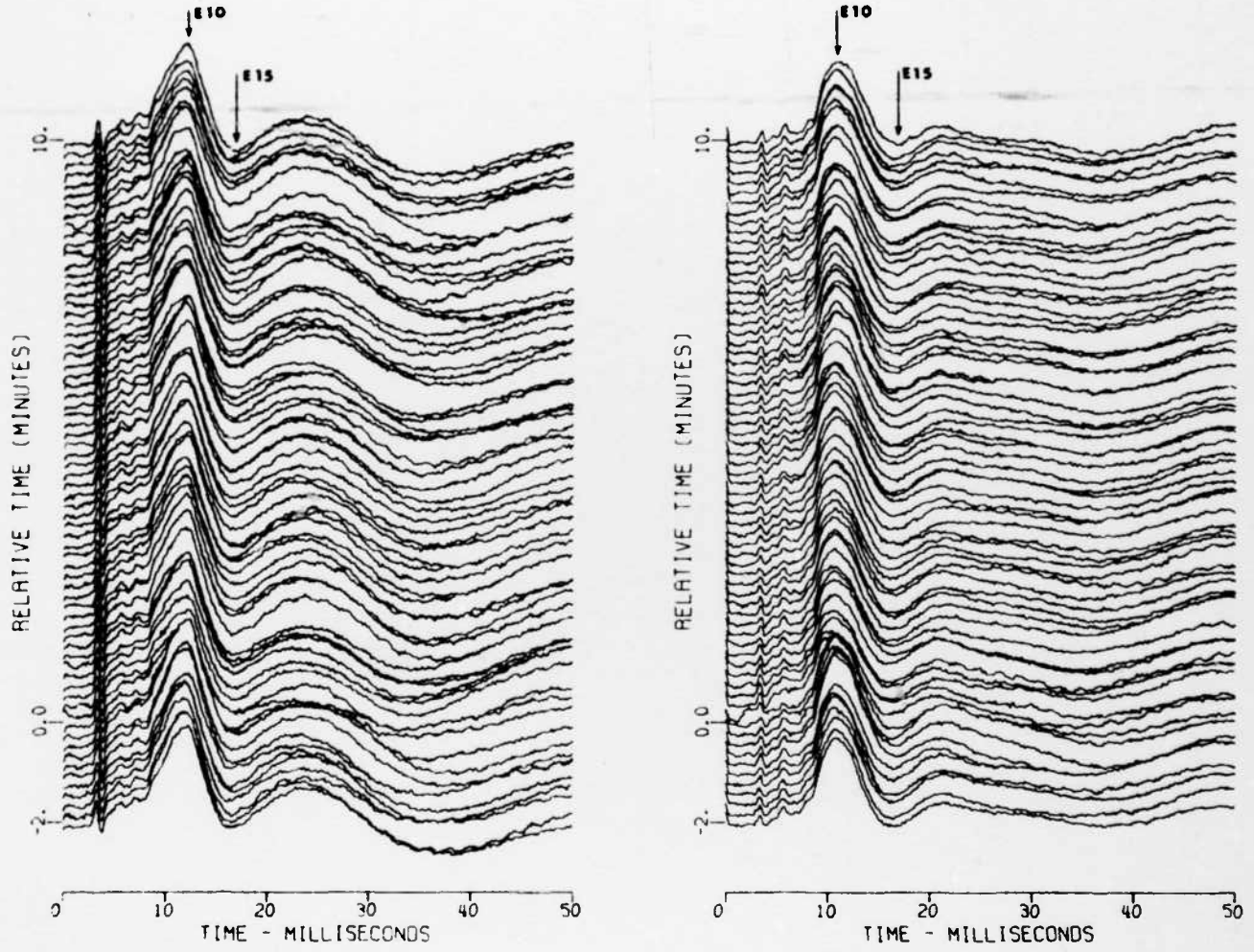
TABLE 4

SUMMARY OF CHANGES IN LATENCY OF E15 COMPONENT

RUN	MEASURE	CXL-LEFT	CXR-RIGHT LEAD
30-G	pre-impact relative latency deviation	1.8%	2.3%
	Maximum change	NS	NS
	Recovery time	0	0
50-G	pre-impact relative latency deviation	1.7%	1.9%
	Maximum change	NS	NS
	Recovery time	0	0
70-G	pre-impact relative latency deviation	0.7%	0.6%
	Maximum change	NS	NS
	Recovery time	0	0
90-G	pre-impact relative latency deviation	2.9%	Note 1
	Maximum change	7.1%	
	Recovery time	40 seconds	

NOTE

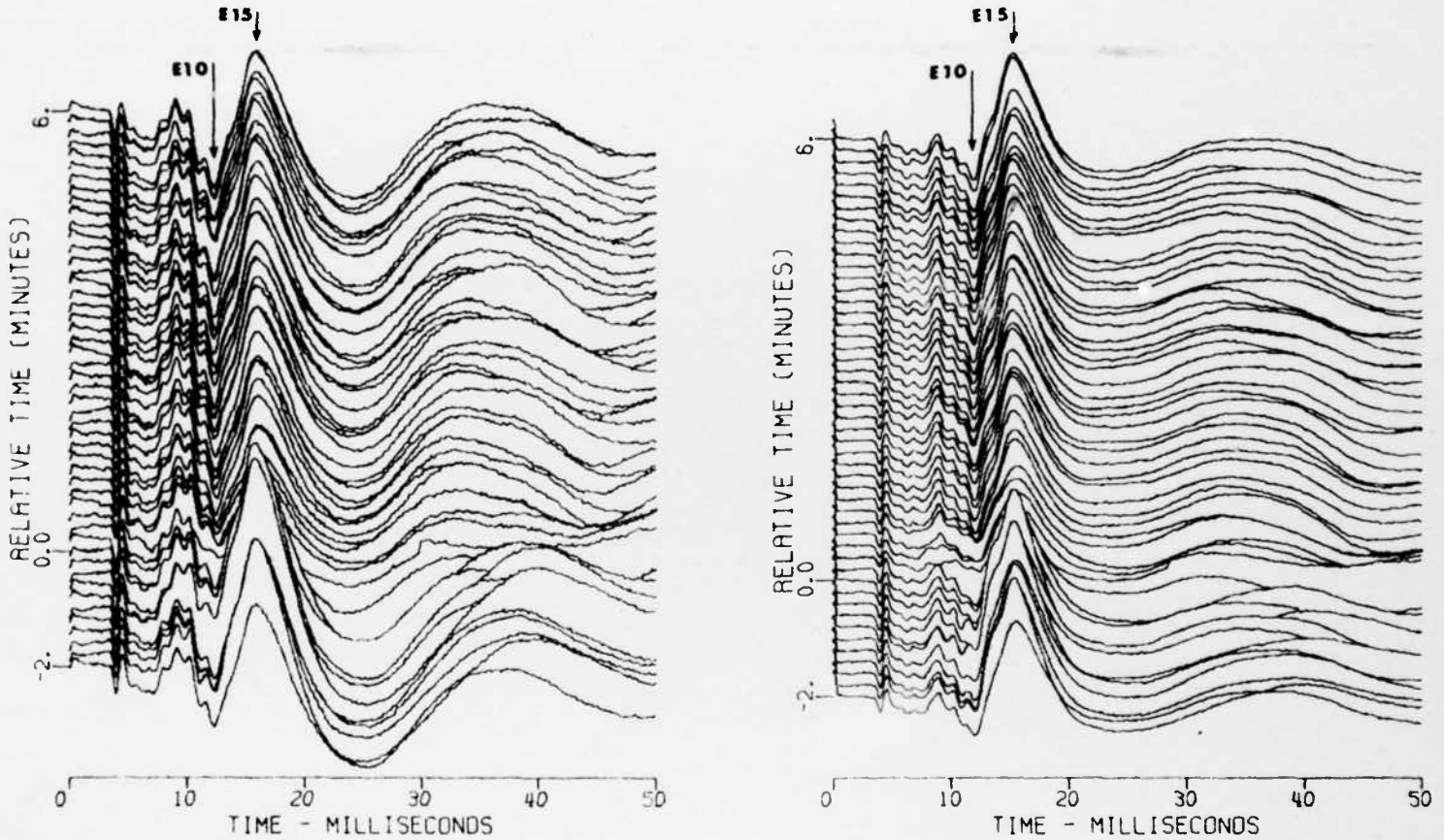
1. The E15 component could not be reliably tracked in the 90-G run.



STIMULUS SITE 7. SPINE RECORDING SITE 3. CXL
LX-3469 30 G - Y 50 RESPONSES AVERAGED

STIMULUS SITE 7. SPINE RECORDING SITE 1. CXR
LX-3469 30 G - Y 50 RESPONSES AVERAGED

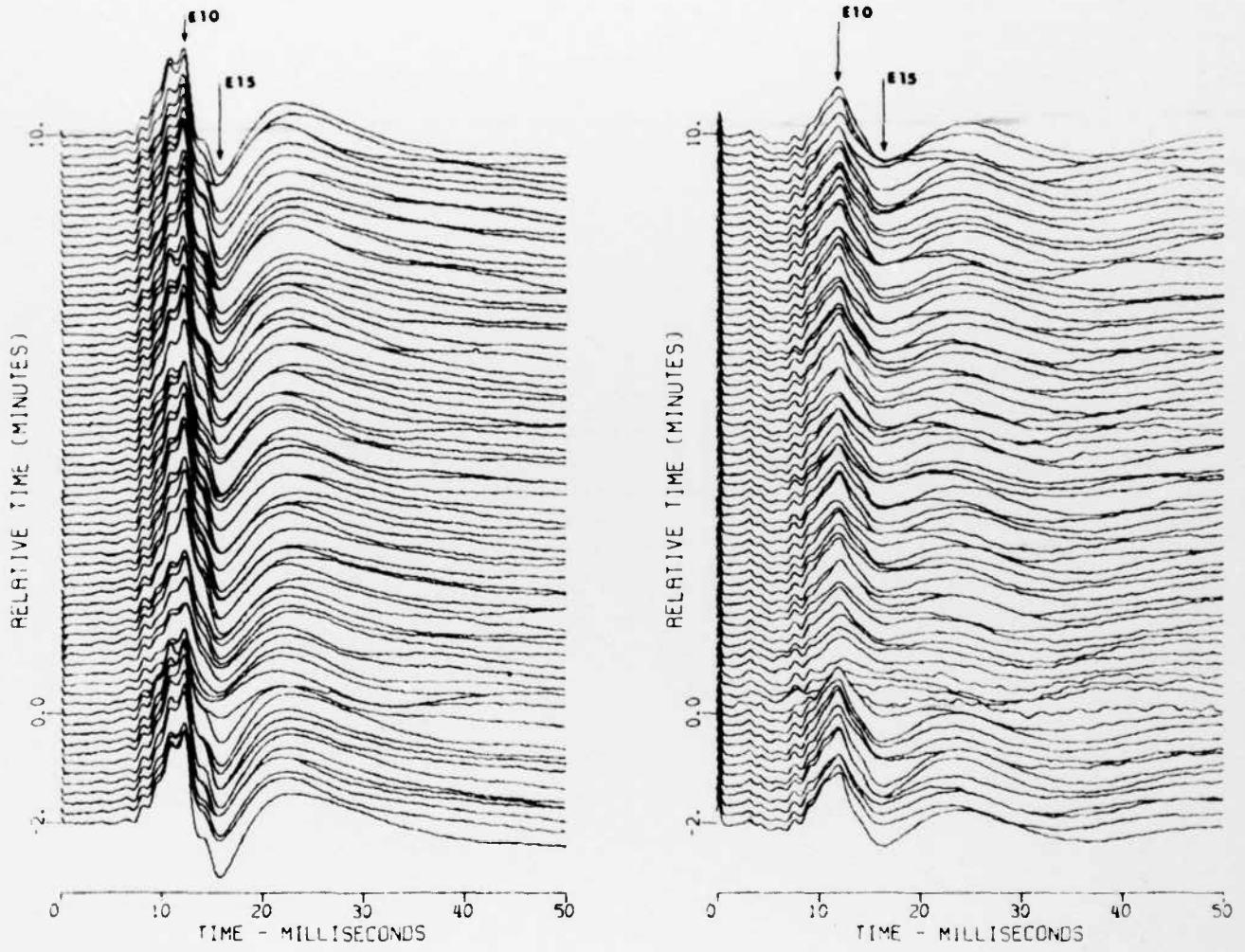
FIGURE 1



STIMULUS SITE 7. SPINE RECORDING SITE 3. CXL
LX-3471 70 G - Y 50 RESPONSES AVERAGED

STIMULUS SITE 7. SPINE RECORDING SITE 1. CXR
LX-3471 70 G - Y 50 RESPONSES AVERAGED

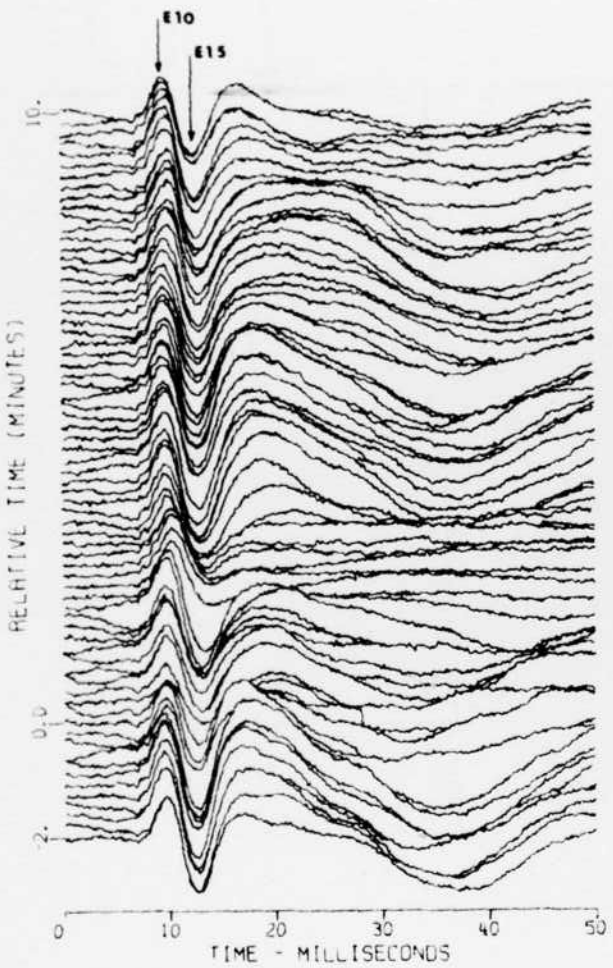
FIGURE 2



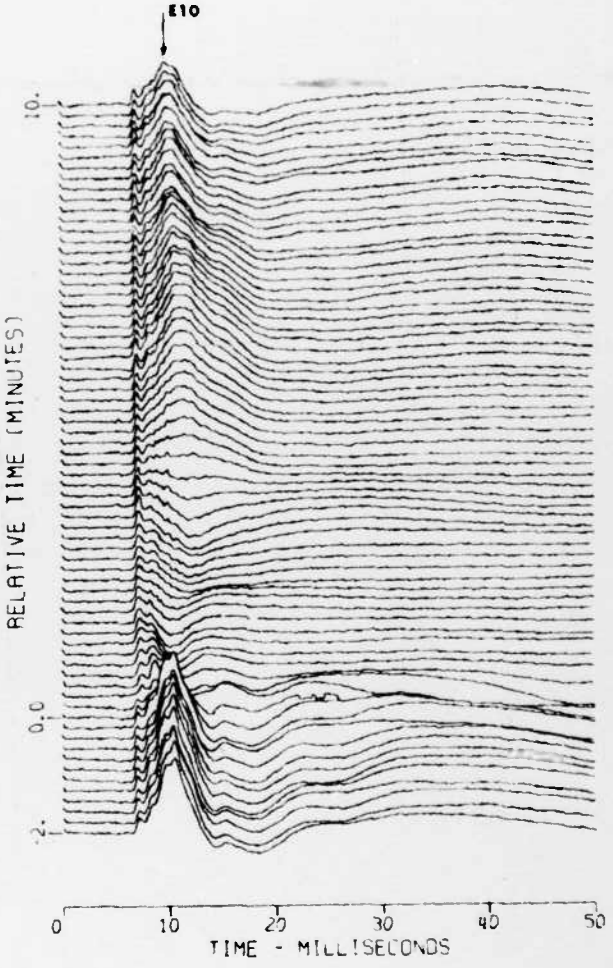
STIMULUS SITE 7. SPINE RECORDING SITE 3. CXL
CX-3473 50 G - Y 50 RESPONSES AVERAGED

STIMULUS SITE 7. SPINE RECORDING SITE 1. CXL
CX-3473 50 G - Y 50 RESPONSES AVERAGED

FIGURE 3

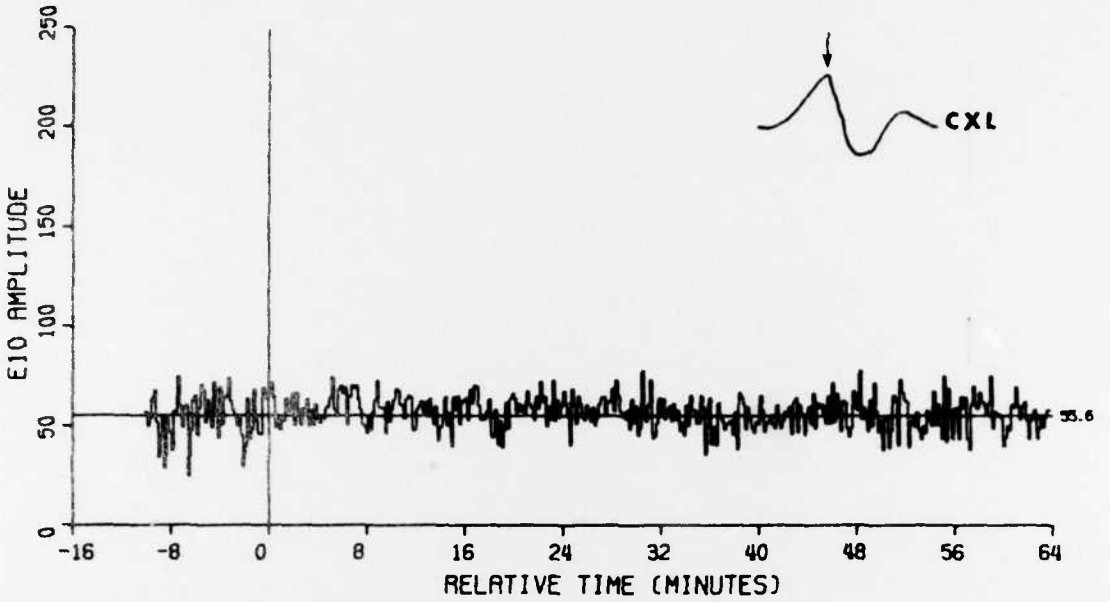


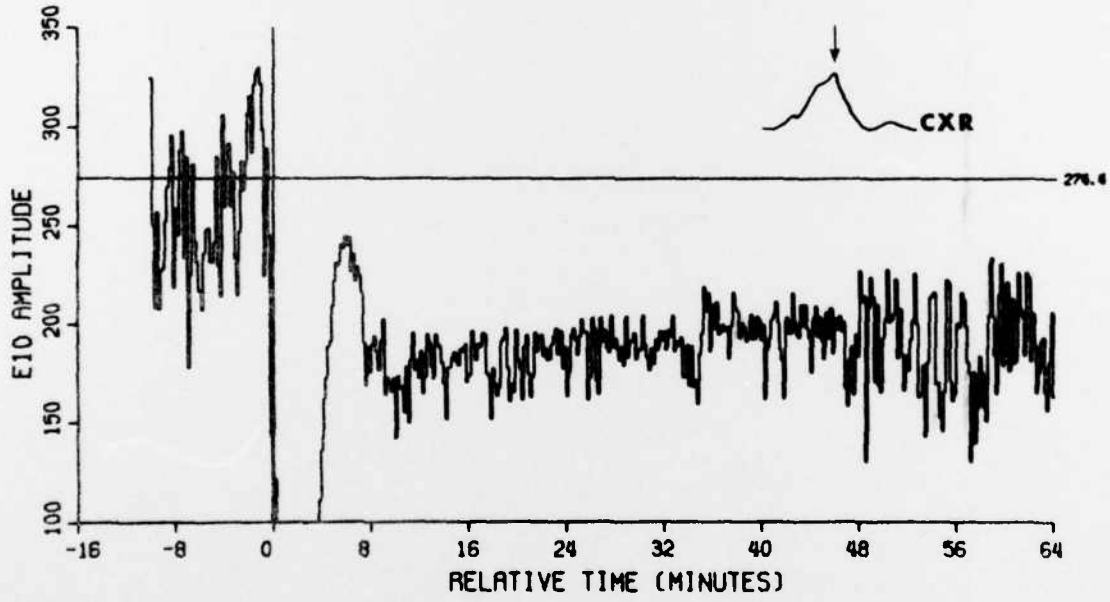
STIMULUS SITE 7. SPINE RECORDING SITE 3. CxL
LX-3475 90 G - Y 50 RESPONSES AVERAGED



STIMULUS SITE 7. SPINE RECORDING SITE 1. CxR
LX-3475 90 G - Y 50 RESPONSES AVERAGED

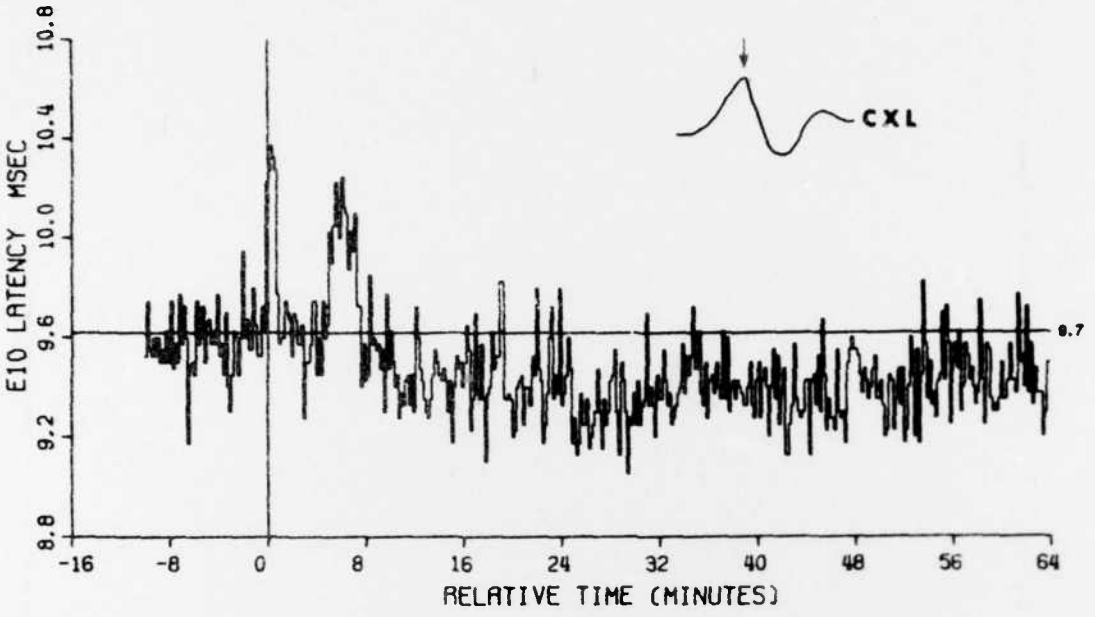
FIGURE 4

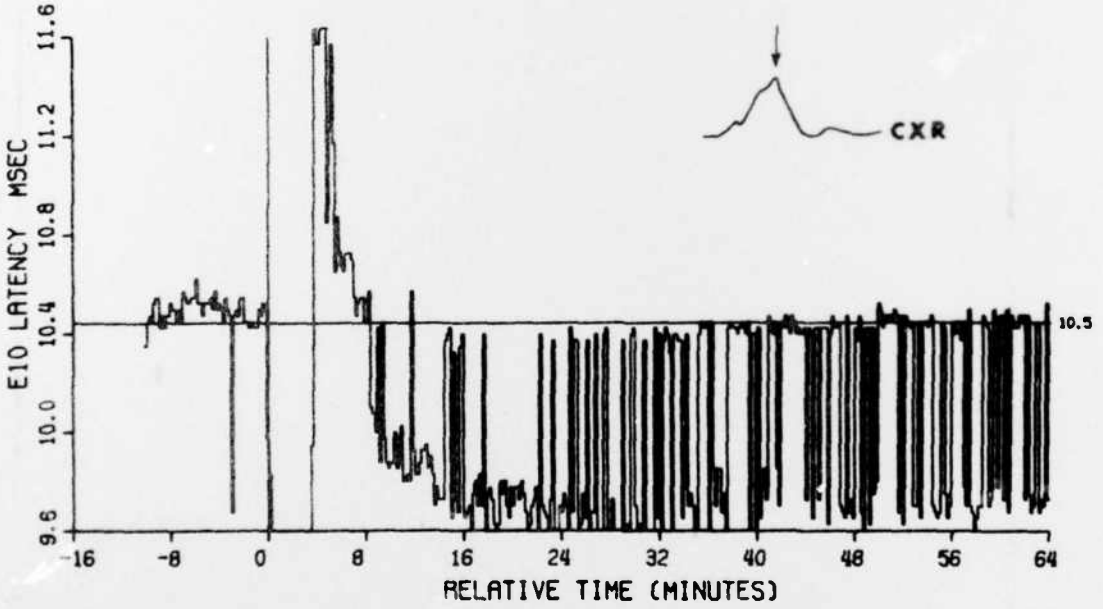




ANIMAL AA-8816 RUN LX-3475 ACCELERATION 90.0 G
STIMULUS SITE 7. SPINE RATE 4.83 /SEC
RECORDING SITE 1. CXR NBR. AVERAGED 50

FIGURE 5





ANIMAL AA-8816 RUN LX-3475 ACCELERATION 90.0 G
STIMULUS SITE 7. SPINE RATE 4.83 /SEC
RECORDING SITE 1. CXR NBR. AVERAGED 50

FIGURE 6

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